Review of Round 2 Subyearling Chinook Tissue Data Report

Prepared by Lyndal Johnson, Environmental Conservation Division, Northwest Fisheries, Science Center, NOAA Fisheries

This report, prepared by Integral Consulting and Windward Environmental for the Lower Willamette Group, contains data on concentrations of metals, butyltins, PCBs, DDTs and other organochlorine pesticides, semi-volatile organic compounds (SVOCs), phenols, phthalates, dioxins and furans, and PAHs, in stomach contents of fish and juvenile salmon from four sites in Portland Harbor. Fish samples were collected from three site within the stuy area, LW2-T01, at river mile 3-4; LW2-T02, at river mile 6-7; LW2-T03, at river mile 9-10, and at an upriver reference sites, LW2-T04, at river mile 17-18.

Various constituents were analyzed using standard EPA methods. Data validation and quality assurance procedures are well documented in the report, and comply with common laboratory standards. Generally, reported data were of high quality, with acceptable detection limits. There were a few cases in which some analytes (e.g., some of the SVOCs and phenols) could not be quantified because of low tissue volume. Also, the number of composite stomach contents samples was low, and made statistical comparison of the data more difficult. These problems are not unexpected, given the small size of the subyearling salmon used for these analyses

Contaminants in stomach contents

As part of this study, contaminants were measured in salmon stomach contents to assess the degree of dietary uptake of toxic compounds in juvenile salmon from the Portland Harbor area. The classes of contaminants measured in salmon stomach contents were more limited than in whole bodies. Metals, phenols, phthalates, and SVOCs were generally not measured. The interpretation below focuses on ∑DDTs, ∑PCBs, and ∑PAHs, because these are the classes of compounds where the best information is available on exposure levels in salmon and toxicity thresholds.

DDTs. The ∑DDT concentrations in stomach contents of juvenile Chinook ranged from 6.4 ng/g ww at site LW2-T04 to 295 ng/g ww at site LW2-T02 (Figure 1). The concentration at site T02 was significantly higher than at either site T01 or the T04 reference site (ANOVA, Tukey-Kramer multiple range test, $p \leq 0.05$; log-transformed values). In comparison to ∑DDT concentrations in stomach contents of salmon sampled from other Pacific Northwest sites (Figure 2), the stomach contents ∑DDT concentrations at sites LW2-T01, T03, and T04 were in the low to moderate range (6.4-10.2 ng/g ww), and were somewhat lower than concentrations reported in juvenile salmon from other sites in the Columbia and Willamette Rivers (35-40 ng/g ww; Johnson et al. 2006ab; Leary et al. 2006). At site LW2-T02, however, concentrations of ∑DDTs in salmon stomach contents were very high, higher than levels in any comparable samples the NWFSC has analyzed from juvenile salmon in Washington or Oregon.

PCBs. Total ∑PCB concentrations in salmon stomach contents ranged from 10.6 ng/g ww in fish from LW2-T04 to 163 ng/g ww in fish from LW2-T03 (Figure 1). The concentration at site T03 was significantly higher than at the T04 reference site, but no other significant differences were observed (ANOVA, $p < 0.05$). The ∑PCB concentration in stomach contents of fish from the T04 reference site was comparable to ∑PCB levels measured in stomach contents of Chinook salmon from rural estuaries in the Pacific Northwest (Figure 3; Johnson et al. 2006ab). At sites T01 and T02, concentrations were in the 58-71 ng/g ww range, comparable to levels found at other sites with some moderate levels of urban development (Johnson et al. 2006). At site T03 stomach contents ∑PCB concentrations were comparable to some of the highest levels observed in salmon from the Pacific Northwest.

PAHs. Total ∑PAH concentrations in stomach contents of juvenile Chinook ranged from from 88 ng/g ww at site LW2-T04 to 2460 ng/g ww at site LW2-T04 (Figure 1), but levels were not significantly different ($p < 0.05$), in part because of the small number of samples analyzed ($1 < n < 2$). In comparison to ∑PAH concentrations in stomach contents of salmon sampled from other Pacific Northwest sites (Figure 4), concentrations at T04, T01, and T03 were low to moderate, while concentrations at site T02 were within the range reported in juvenile salmon from other urban sites. However, concentrations at all sites were below estimated thresholds for ∑PAH effects on growth, metabolism, and immune function of about 7,000 ng/g ww or above based on laboratory studies (Meador et al. 2006; Palm et al. 2003), or levels observed in the field-collected fish where these types of problems have been documented (~5000 ng/g ww; Arkoosh et al. 2002; Casillas et al. 1998).

Contaminants in Salmon Whole Bodies

Lipid content. Lipid content of fish bodies ranged from 1.5-1.9%. These levels are very similar to lipid levels the NWFSC has measured in juvenile salmon from other Columbia River and Willamette sites (Johnson et al. 2006a,b, Leary et al. 2006), and within the typical range reported for this species (Beckman et al. 2002).

Metals. Metals measured in salmon whole bodies included aluminum, antimony, arsenic, cadmium, chromium, copper, lead, mercury, nickel, selenium, silver, and zinc. The metals detected at the highest concentrations were aluminum and zinc. Concentrations of Al ranged from 3-13 mg/kg wet wt, while concentrations of Zn ranged from 24-31 mg/kg wet wt. However, there was no evidence of elevated concentrations in fish from sites within Portland Harbor as compared to the reference area. Concentrations of all other metals were $\leq$ 1 mg/kg wet wt, and in many cases < 0.1 ng/g ww. Like Zn and Al, concentrations of all other metals were very similar in whole bodies of salmon from all of the sampling sites, and concentrations in salmon from sites T01, T02, and T03 showed little difference from those in fish from the T04 reference site. Moreover, concentrations of As, Cd, Hg, Pb, Se, and Zn were all similar to or below concentrations reported for resident fish in the Columbia River by Hinck et al. 2006.

Concentrations of metals appeared to be below toxicity thresholds, where such data are available. According to Hinck et al. 2006, whole body concentrations of As, Cd, and Zn were below toxicity thresholds in resident fish they sampled, and concentrations in the juvenile salmon from the Willamette were at or below concentrations reported by Hinck et al. (2006). For Hg, lowest reported toxicity thresholds are 0.3 -0.7 ug/g ww for impacts on feeding efficiency, competitive ability, and other behavioral endpoints (Fjeld et al. 1998; Kania and O'Hara 1974). At 0.011 to 0.013 ug/g ww, whole body total mercury concentrations in Willamette River salmon were below these levels for all samples. For Se, toxic effects have been reported at whole body concentrations as low as 1 ug/g ww (Lemly 1996, 2002). Again, the Willamette salmon were well below this level with concentrations ranging from 0.18-0.27 ug/g ww. As for Pb, reduced hatching success has been documented in concentrations of 0.4 ug/g ww in eggs and reduced growth in various life stages at concentrations of 4-8 ug/g ww (Holcombe et al. 1976). Levels in whole bodies of sampled salmon were well below these levels, ranging from 0.03 to 0.07 ug/g ww.

PAHs. Because PAHs are extensively metabolized by fish, high concentrations of these compounds in whole body samples were not expected. However, low but measurable concentrations of PAHs, primarily low molecular weight compounds, were found, at concentrations ranging from 5-19 ng/g ww. Mean concentrations were about twice as high in salmon from sites T01, T02, and T03 than in fish from the reference site, suggesting greater exposure to PAHs in Portland Harbor fish. However, even in the Portland Harbor fish, whole body PAH levels were below those we observed in pre-release hatchery fish from several Columbia River hatcheries (25-41 ng/g ww) as part of a monitoring study conducted with USGS and LCREP (Leary et al. 2006).

PCBs. On a wet weight basis, whole body PCB concentrations ranged from 12.1 ng/g ww at the T04 reference site to 253 ng/g ww at site T03 (Figure 4). When adjusted for lipid content, the average concentrations ranged from 868 ng/g lipid at site T04 to 11,300 ng/g lipid at site T03 (Figure 4). Both wet wt and lipid wt PCB concentrations were significantly higher at sites T01, T02, and T03 than at the T04 reference site (ANOVA, Tukey-Kramer multiple range test, $p \leq 0.05$, log-transformed values). In comparison with ∑PCB concentrations in juvenile salmon from other Pacific Northwest sites, levels in fish from sites T01, T02, and T03 were among the highest reported (Figure 5). Moreover, mean lipid adjusted PCB concentrations in fish sampled from all sites except T04 were above the effect threshold of 2400 ng/g lipid proposed by Meador et al. (2002). This suggests that there is a substantial risk of delayed mortality, immune dysfunction, and other health problems in salmon from these sites due to PCB exposure.

DDTs. Mean concentrations of DDTs in whole body salmon samples range from 9 ng/g wet wt at site T04 to 247 ng/g wet wt range at site T02 (Figure 6). On a lipid weight basis, they ranged from 492 ng/g lipid at T04 to 14,800 ng/g lipid at T02 (Figure 6). Both wet wt and lipid wt DDT concentrations were significantly higher at sites T01, T02, and T03 than at the T04 reference site (ANOVA, Tukey-Kramer multiple range test, $p \leq 0.05$, log-transformed values). Moreover, both wet wt and lipid wt DDT concentrations were significantly higher at site T02 than at sites T01 or T03. Compared to other data collected by NWFSC, the DDT concentration in salmon from site T02 was among the highest observed (Figure 7).

Toxic effects of DDTs have been reported at concentrations as low as 500 ng/g ww in some fish species (Jarvinin and Ankley 1999) and Beckvar et al. (2005) has proposed a critical tissue value of 600 ng/g wet wt for effects of ∑DDTs on adult and juvenile fish, including salmonids. Even in salmon from site T02, tissue concentrations of DDTs were below these levels. However, these toxicity thresholds are not adjusted for lipid contents, which can have a strong influence on the toxicity of bioaccumulative contaminants (Elskus et al. 2005; Lassiter and Hallam, 1990). The typical lipid content of laboratory-reared salmonids, which were used to generate much of the toxicity data used in analyses such as Beckvar et al. (2005), is 8-10% (Meador et al. 2002), where as in the juvenile salmon collected in the present study, the lipid content was only 1-2%. If we assume a 10% lipid content for salmonids used to generate the 600 ng/g ww toxicity threshold, the lipid adjusted value would be 6000 ng/g lipid. In salmon from sites T01, T03, an T04, lipid-adjusted ∑DDT concentrations are all below this level, but fish from site T02 exceed it. Fish from site T02 might also be a threat to piscivorous wildlife, as ∑DDTs exceed proposed the fish tissue criteria for wildlife of 200 ng/g wet wt (Newell et al. 1987).

Butyltins. Butyltin concentrations in salmon whole bodies ranged from 1-2 ng/g ww in fish from T04 to 8-9 ng/g ww in fish from T02. Effects of TBT on growth of salmonids has been reported at tissue concentrations of 2000 ng/g dry wt or about 400 ng/g wet wt (Seinen et al. 1981; DeVries et al. 1991), far above concentrations observed in the Willamette salmon.

SVOCs. These compounds, which included di-, tri-, and hexachlorobenzens, benzyl alchohol, dibenzofuran, hexachlorobenzene, hexaclorobutadiene, hexachloroethane, and N-nitrosodiphenylalanine, were generally below quantifiable limits in the salmon sample collected in this study. Estimated concentrations of HCB and HCBD were typically < 1 ng/g ww, while concentrations of the other compounds range from 20-130 ng/g ww. Benzyl alcohol was detected in fish from T01 and T03 at concentrations in the 55-200 ng/g ww range. In several whole body samples, concentrations of SVOC could not be measured reliably because of small sample size.

Phenols. Like the SVOC, these compounds were generally below detection limits or not quantifiable in the salmon whole body samples. Estimated concentrations range from 25-100 ng/g ww at T01 and T04, up to 140-700 ng/g ww at sites T02 and T03, but still below limits of detection, suggesting these compounds could not be reliably measured. Phenol and pentachlorophenol were found at quantifiable levels in one samples from T01, with concentrations of 38 ng/g ww and 140 ng/g ww, respectively. NOAA Fisheries has proposed a tissue benchmark of 300 ng/g ww for chlorinated phenolic compounds for salmonids (NMFS 2004), and some of the estimated concentrations in fish from the Portland Harbor sites were above this level. Although estimated values may be inaccurate, potential toxicity from chlorinated phenolic compounds cannot be ruled out in fish from these sites.

Other OC pesticides. These compounds were generally below detection limits or at low concentrations (< 1 ng/g ww). Detection limits for toxaphene were quite high (up to 300 ng/g ww), suggesting that this compounds could not be reliably quantified in these samples.

Dioxins and Furans. Impacts of dioxins and furans are somewhat difficult to evaluate because the analysis doesn't include calculation of TEQs. I attempted to calculate TEQs for these compounds using toxic equivalency factors (TEFs) of Van den Berg et al. (1998). According to these estimates, the TCDD/TCDF TEQs for fish samples from sites T01 to T04 are well below the NOAA and EPA guideline of 9 pg/g ww (NMFS 2004). The highest value calculated was 2.3 pg/g ww for site T03. Values for the other sites were < 1 pg/g ww.

Summary

Overall, the data in this report indicate significant exposure of juvenile Chinook salmon from Portland Harbor to several major classes of contaminants. Concentrations of PCBs and DDTs were both significantly higher in whole bodies of salmon from sites T01, T02, and T03 in Portland Harbor than in salmon from the T04 reference site, where whole body contaminant concentrations were low, similar to concentrations observed at rural estuaries from other sites in the Pacific Northwest (Johnson et al. 2006). Differences were less pronounced for concentrations of these contaminants in stomach contents, in part because of the small number of sample replicates available for analysis. However, contaminant concentrations were generally higher in stomach contents of salmon from the three Portland Harbor sites than in salmon from the reference site. When contaminant concentrations in salmon whole bodies and stomach contents from the three Portland Harbor sites were compared with other Pacific Northwest sites, concentrations were generally within the ranges reported for other urban areas (Johnson et al. 2006ab, Leary et al. 2006). Concentrations of DDTs in fish from site T02, and concentrations of PCBs in fish from sites T02 and T03, were among the highest reported. PAHs were also detected in stomach contents and whole bodies of salmon from the Portland Harbor sites, but at more moderate levels. Generally there was a positive relationship between contaminants in stomach contents and contaminants in whole bodies, and body burdens were clearly elevated in juvenile salmon from Portland Harbor in comparison to the upstream reference site, indicating that fish are remaining in the Portland Harbor long enough to accumulate contaminants.

When concentrations of contaminants in Portland Harbor salmon were compared with available tissue residue guidelines (Meador et al. 2002; Beckvar et al. 2005), levels of DDTs and PCBs appeared to be high enough in fish from some sites to cause toxicity. Concentrations of PAHs in stomach contents of salmon from all sites were below concentrations that have been associated with impacts on immune function and growth in field and laboratory studies (Arkoosh et al. 2001, 2004; Casillas et al. 1998, Palm et al. 2003; Meador et al 2006), but could contribute to toxicity in conjunction with other contaminants.

Concentrations of other classes of contaminants were generally similar at Portland Harbor and reference sites, and relatively low in comparison to values reported in the literature for other fish species (e.g., Hinck et al. 2006). A possible exception are the chlorinated phenols, which could not be measured reliably in most samples, but whose estimated concentrations exceeded NOAA's recommended tissue benchmark.

In addition to the contaminants measured in this study, it might be useful to analyze for the fire retardants, polybrominated diphenyl ethers (PBDEs), emerging contaminants that may affect thyroid function and neural development (Siddiqi et al. 2003). In preliminary data from recent study conducted by the NWFSC in collaboration with the Lower Columbia River Estuary Partnership (Leary et al. 2006), concentrations of PBDEs as high as 99 ng/g ww were observed in whole bodies of salmon from Morrison Street Bridge. This concentration is substantially higher than the majority of fish samples analyzed in a survey of PBDEs conducted by Washington DOE (Johnson et al. 2006c), suggesting these contaminants may be a problem in the Portland area.

References

Arkoosh MR, Casillas E, Clemons E, Huffman P, Kagley AN, Collier TK, Stein JE. Increased susceptibility of juvenile chinook salmon (*Oncorhynchus tshawytscha*) to vibriosis after exposure to chlorinated and aromatic compounds found in contaminated urban estuaries. J. Aquat. Anim. Health 2001; 13:257-268.

Arkoosh MR, Casillas E, Huffman P, Clemons E, Evered J, Stein JE, Varanasi U. Increased susceptibility of juvenile chinook salmon (Oncorhynchus tshawytscha) from a contaminated estuary to the pathogen Vibrio anguillarum. Trans. Am. Fish. Soc. 1998; 127:360-374.

Beckman, B. R., W. W. Dickhoff, W. S. Zaugg, C. Sharpe, S. Hirtzel, R. Schrock, D. A. Larsen, D. Ewing, A. Plamisano, C. B. Schreck, C. V. Mahnken. 1999. Growth, smoltification, and smolt-to-adult return of spring chinook salmon (*Oncorhynchus tshawytshcha*) from hatcheries on the Deschutes River, Oregon. Trans. Am. Fish. Soc. 1999;128:1125-1150.

Beckvar N, Dillon TM, Read LB. 2005. Approaches for linking whole-body fish residues of mercury or DDT to biological effects thresholds. Environ. Toxicol. Chem. 24:2094-2105.

Biro PA, Morton AE, Post JR, Parkinson EA. Over-winter lipid depletion and mortality of age-0 rainbow trout (*Oncorhynchus mykiss*). Can. J. Fish. Aquat. Sci. 2004; 61:1513-1519.

Casillas E, Eberhart B-TL, Sommers FC, Collier TK, Krahn MM, Stein JE. 1998. Effects of Chemical contaminants from the Hylebos Waterway on growth of juvenile chinook salmon. Interpretive Report prepared for NOAA Damage Assessment Center.

de Vries H, Penninks AH, Snoeij NJ, Seinen W. 1991. Comparative toxicity of organotin compounds to rainbow trout (Oncorhynchus mykiss) yolk sac fry. Science of the Total Environment 103: 229–243.

Elskus A, Collier TK, Monosson E. Interactions between lipids and persistent organic pollutants in fish. Pages 119-152 in Environmental Toxicology, Elsevier, San Diego; 2005.

Fjeld, E. TO Haugan, and LA Vollestad. 1998. Permanent impairment in the feeding behavior of grayling (*Thymallus thymallus*) exposed to methylmercury during embryogenesis. Sci. Total Environ. 213-247-254.

Hinck JA, Schmitt CJ, Blazer VS, Denslow ND, Bartish TM, Anderson, PJ, Coyle JJ, Dethloff, GM, Tillitt DE. 2006. Environmental contaminants and biomarker responses in fish from the Columbia River and its tributaries: Spatial and temporal trends. Sci. Total Environ. 366:549-578.

Holcombe, GW, DA Benoit, EN Leonard, and JM McKim. 1976. Long-term effects of lead eposure on three generations of brook trout (*Salvelinus fontinalis*). J. Fish. Res. Board Can. 33:1731-1741.

Jaravenin, AW and GT Ankley. 199. Linkage of effects to tissue residues: development of a comprehensive database for aquatic organisms exposed to organic and inorganic chemicals. Pensacola FL, Society of Environmental Toxicology and Chemistry (SETAC) Press. 364 pp.

Johnson LL, Ylitalo GM, Arkoosh MR, Kagley AN, Stafford C, Bolton JL, Buzitis J, Anulacion BF, Collier TK. 2006a. Contaminant exposure in outmigrant juvenile salmon from Pacific Northwest estuaries. Environ. Monitor. Assess. 2006; (in press).

Johnson, L.L., G.M. Ylitalo, C.A. Sloan, B.F. Anulacion, A.N. Kagley, M.R. Arkoosh, and T.K. Collier. 2006b. Persistent Organic Pollutants in Outmigrant Juvenile Chinook Salmon from the Lower Columbia Estuary. Sci. Total Environ. (in review).

Johnson, A., K. Seiders, C. Deligeannis, K. Kinney, P. Sandvik, B. Era-Miller, and D. Alkire. 2006c. PBDE Flame Retardants in Washington Rivers and Lakes: Concentrations in Fish and Water, 2005-06. Publication No. 06-03-027. Washington State Department of Ecology, Olympia, Washington

Kania, HJ, and J. O’Hara. 1974. Behavioral alterations in a simple predator-prey system due to sublethal exposure to mercury. Trans. Am. Fish. Soc. 103:134-136.

Lassiter RR, Hallam TG. Survival of the fattest: implications for acute effects of lipophilic chemicals on aquatic populations. Environ. Toxicol. Chem. 1990; 9: 585–595.

Leary, JC, JL. Morace, CA Simenstad, JL. Burke, TD Counihan, JR Hatten, IR Waite, KL Sobocinski, J. Dietrich, J. Spromberg, L. Johnson, and G. Ylitalo. 2006. Lower Columbia River Ecosystem Monitoring Project Annual Report for Year 3 (September 2005 to August 2006). Lower Columbia River Estuary Partnership, Portland Oregon.

Lemly, A.D. 1996. Assessing the toxic threat of selenium to fish and aquatic birds. Environ. Monit. Assess. 43:19-35.

Lemly, AD. 2002. Selenium Assessment in Aquatic Ecosystems. New York, NY. Springver verlag. 160 pp.

Meador JP, Collier TK, Stein JE. Use of tissue and sediment-based threshold concentrations of polychlorinated biphenyls (PCBs) to protect juvenile salmonids listed under the US Endangered Species Act. Aquat. Conserv. Mar. Freshw. Ecosyst. 2002; 12: 493–516.

Meador JP, Sommers FC, Ylitalo GM, Sloan CA. Altered growth and physiological responses in juvenile chinook salmon (*Oncorhynchus tshawytshca*) from dietary exposure to polycyclic aromatic hydrocarbons (PAHs). Can. J. Fish. Aquat. Sci 2006; 63:2364-2376.

Newell, AJ, DW Johnson, and LK Allen. 1987. Niagra River biota contamination project: fish flesh criteria for piscivorous wildlife. Technical report 87-3. Albany: New York Department of Environmental Conservation, Dividiosn of Fish and Wildlife, Bureau of Environmental Protection. 182 pp.

NMFS (National Marine Fisheries Service). 2004. Endangered Species Act Section 7 Consultation Biological Opinion and Magnuson-Stevens Fishery Conservation and Management Act Essential Fish Habitat Consultation for United States Environmental Protection Agency on Potlatch Pulp and Paper Mill National Pollution Discharge and Elimination System Permit.

Palm, R.C. Jr., Powell, D.B., Skillman, A., and Godtfredsen, K. 2004. Immunocompetence of juvenile chinook salmon against *Listonella anguillarum* following dietary exposure to polycyclic aromatic hydrocarbons. Environ. Toxicol. and Chem. 22:2986–2994.

Seinen W, T Helder, H Vernij, A Penninks, and P Leeuwangh . 1981. Short term toxicity of tri-n-butyltin chloride in rainbow trout (*Salmo gairdneri*) yolk sac fry. Sci. Total Environ. 19: 155–166.

Siddiqi MA, Laessig RH, Reed KD. 2003. Polybrominated diphenyl ethers (PBDEs): new pollutants-old diseases. Clin Med Res. 1:281-90.

Van den Berg, M., Birnbaum, L., Bosveld, B.T.C., Brunstrom, B., Cook, P., Feeley, M., Giesy, J.P., Hanberg, A., Hasegawa, R., Kennedy, S.W., Kubiak, T., Larsen, J.C., van Leeuwen, F.X.R., Liem, A.K.D., Nolt, C., Peterson, R.E., Poellinger, L., Safe, S., Schrenck, D., Tillitt, D., Tysklind, M., Younes, M., Waern, F., and Zacharewski, T. (1998). Toxic Equivalency Factors (TEFs) for PCBs, PCDDs, PCDFs for Humans and for Wildlife. Environmental Health Perspectives 106:775.

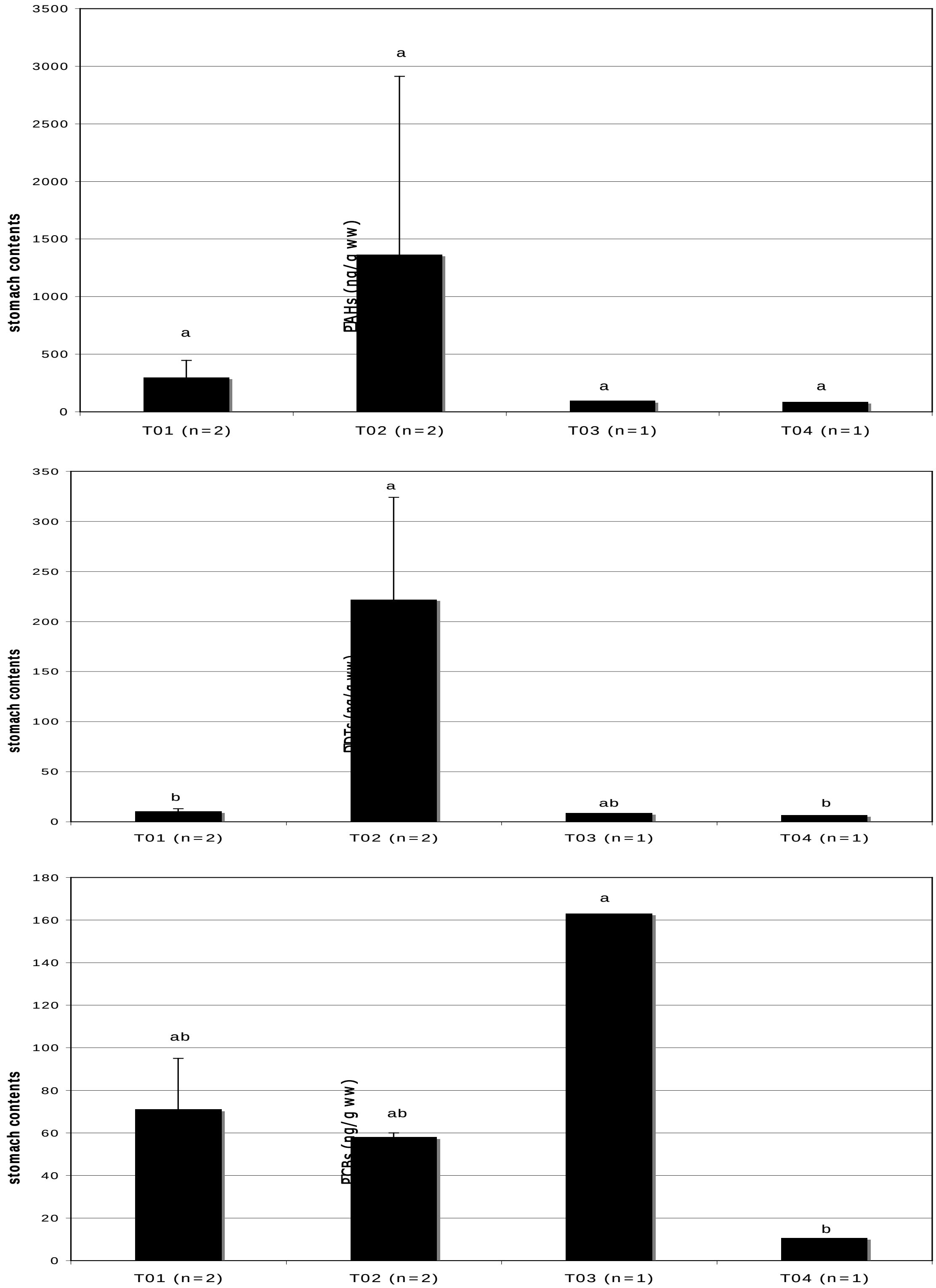


Figure 1. Concentrations of PCBs, DDTs, and PAHs (ng/g wet wt) in stomach contents of juvenile salmon from the Willamette River. Mean values with different letter designations are significantly different (1-way ANOVA, Tukey-Kramer multiple range test, $p \leq 0.05$; log-transformed values).

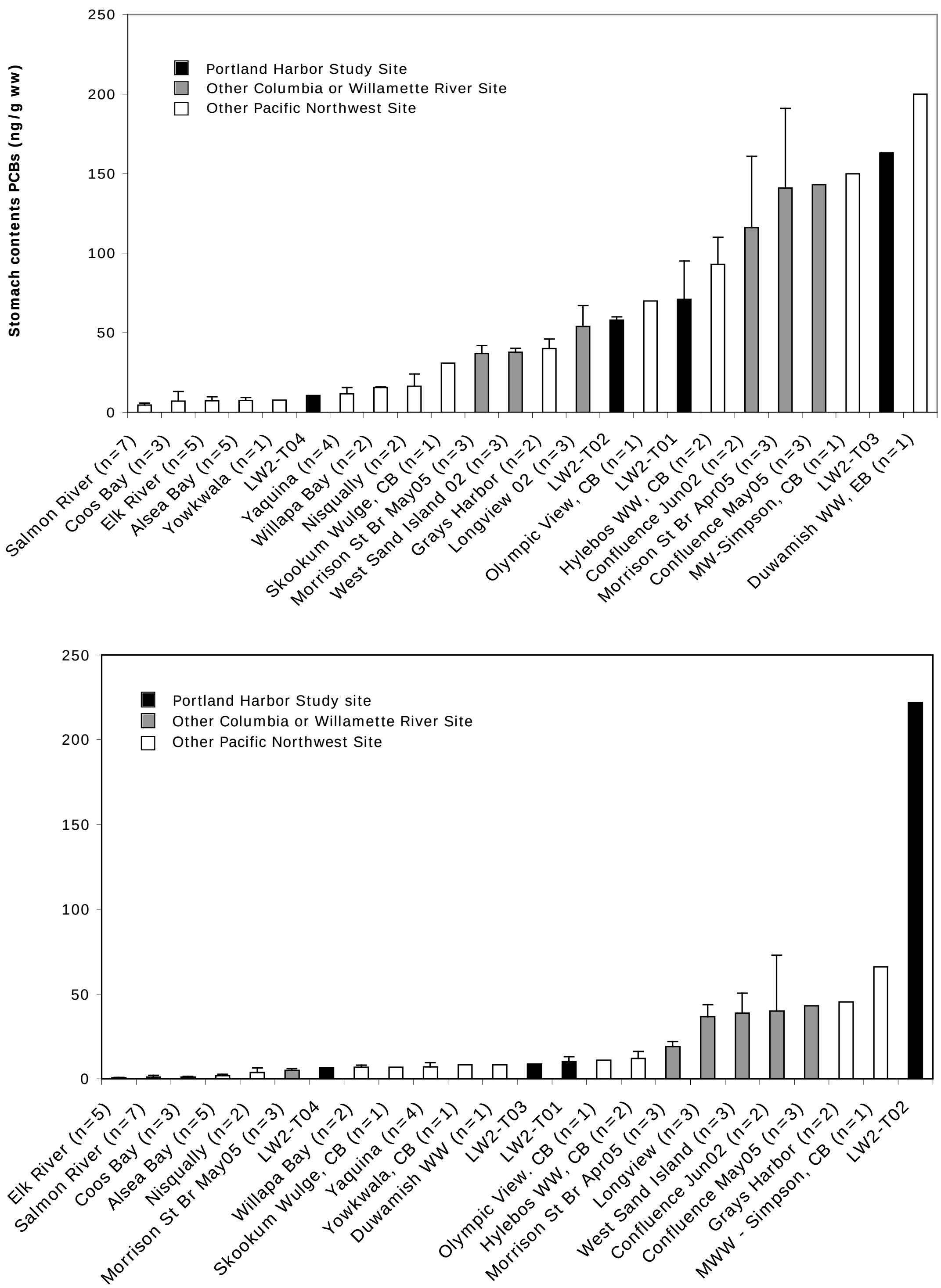


Figure 2. Concentrations of ∑PCBs and ∑DDTs in stomach contents of juvenile salmon from the Willamette River as compared other Pacific Northwest sites. Data from Johnson et al. 2006a,b; Leary et al. 2006).

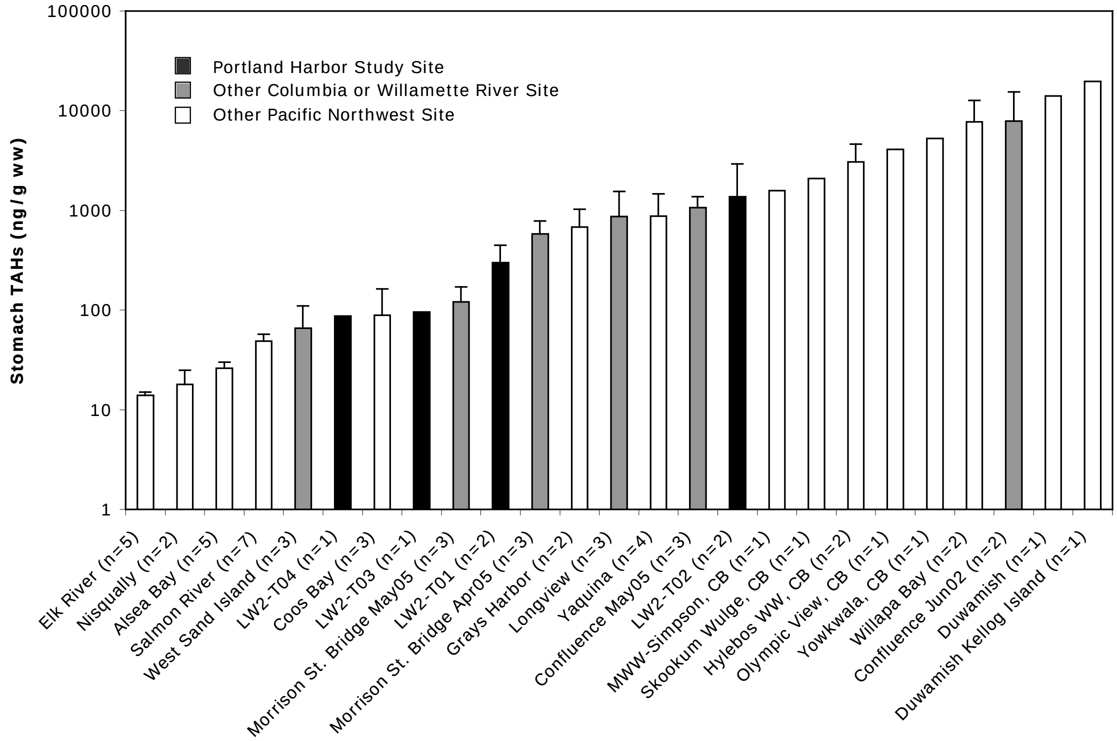


Figure 3. Concentrations of ∑PAHs in stomach contents of juvenile salmon from the Willamette River as compared other Pacific Northwest sites. Data from Johnson et al. 2006a,b; Leary et al. 2006).

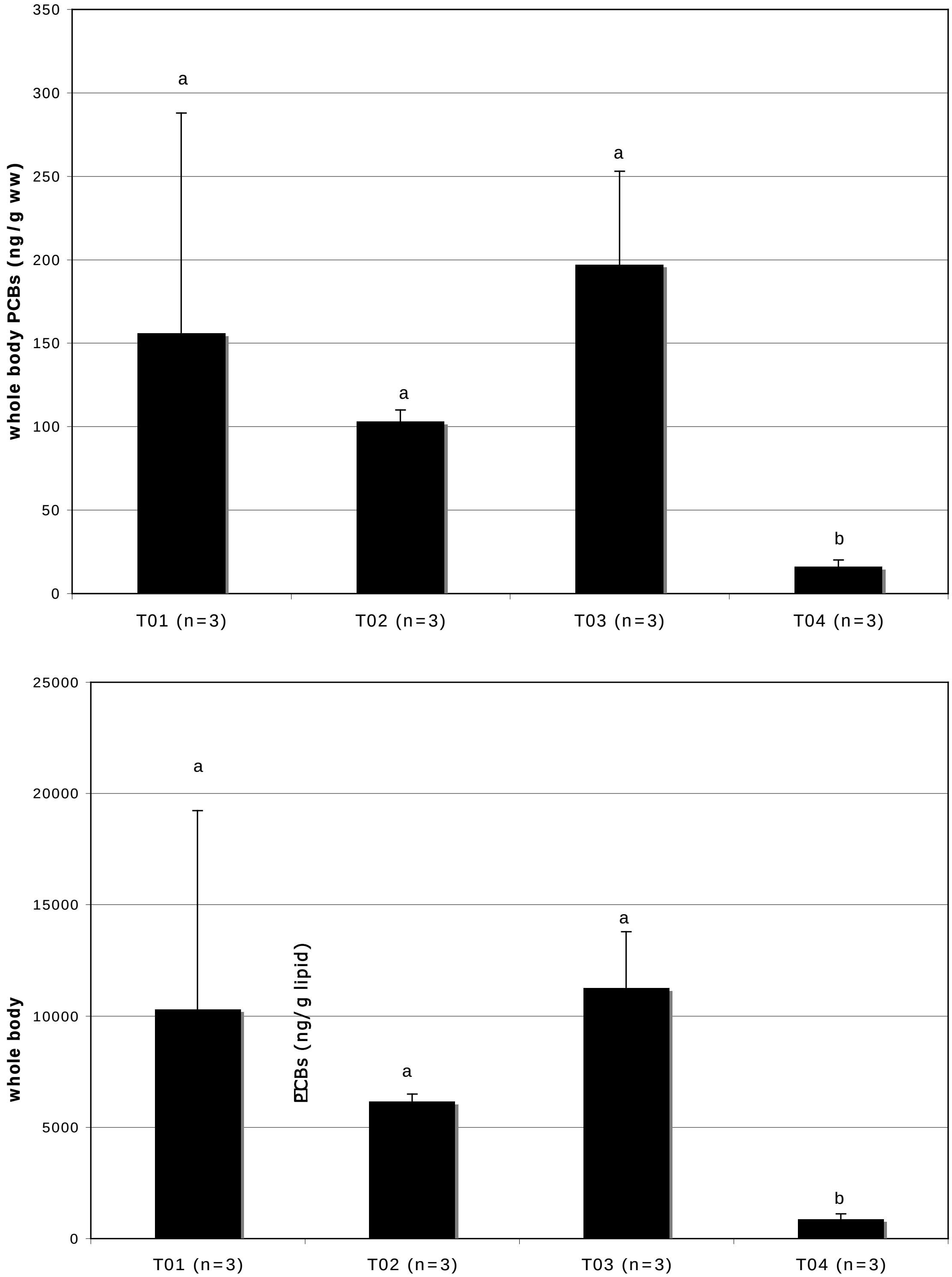
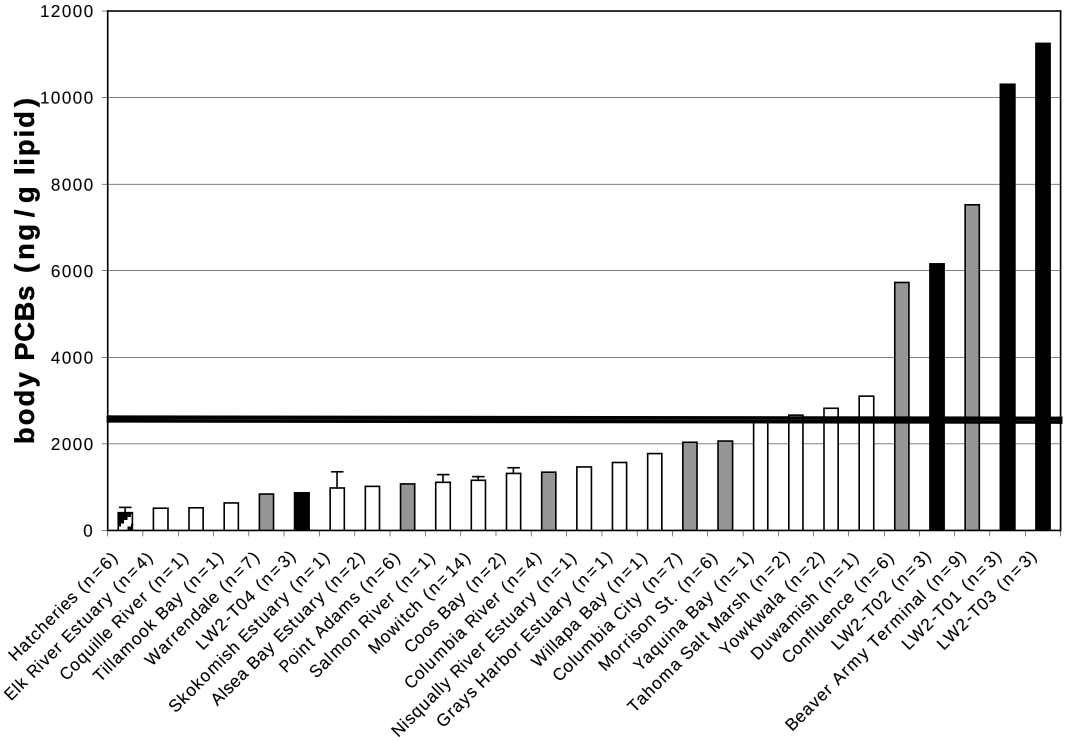


Figure 4. Concentrations of ∑PCBs (ng/g ww and ng/g lipid) in whole bodies of juvenile salmon from Willamette River sites. Mean values with different letter designations are significantly different (1-way ANOVA, Tukey-Kramer multiple range test, $p \leq 0.05$; log-transformed values).



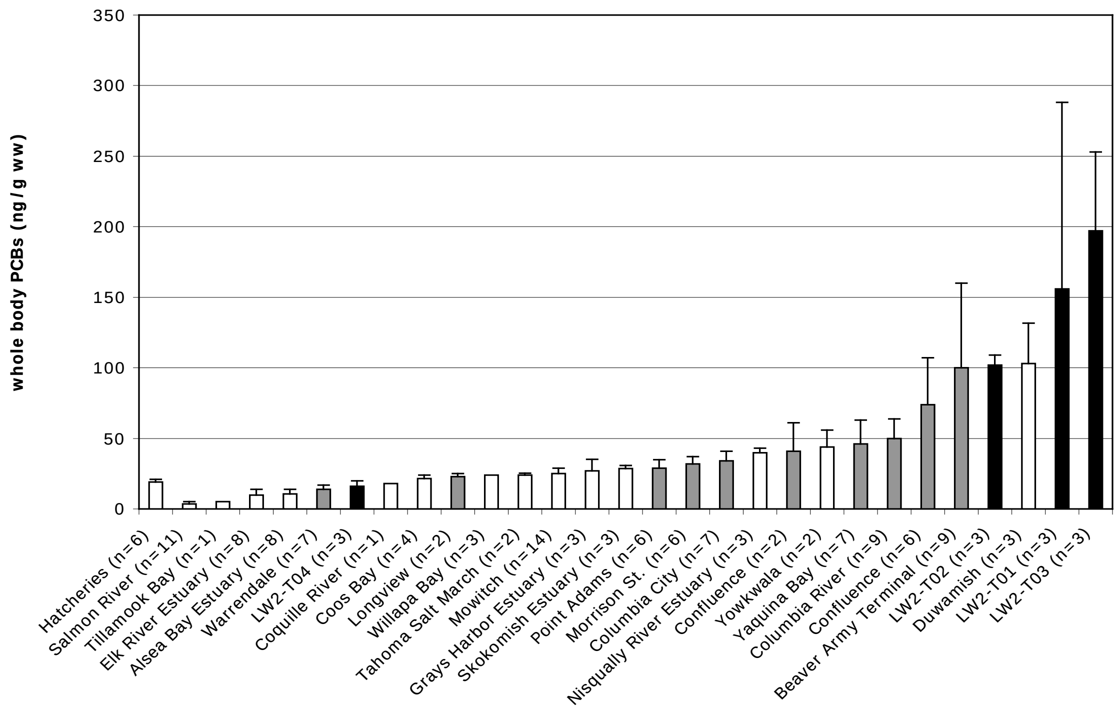


Figure 5. Concentrations of PCBs (ng/g lipid and ng/g wet wt) in whole bodies of juvenile salmon from the Willamette River as compared other Pacific Northwest sites. Data from Johnson et al. 2006a,b; Leary et al. 2006). Solid line indicates estimated toxicity threshold, based on Meador et al. 2002.

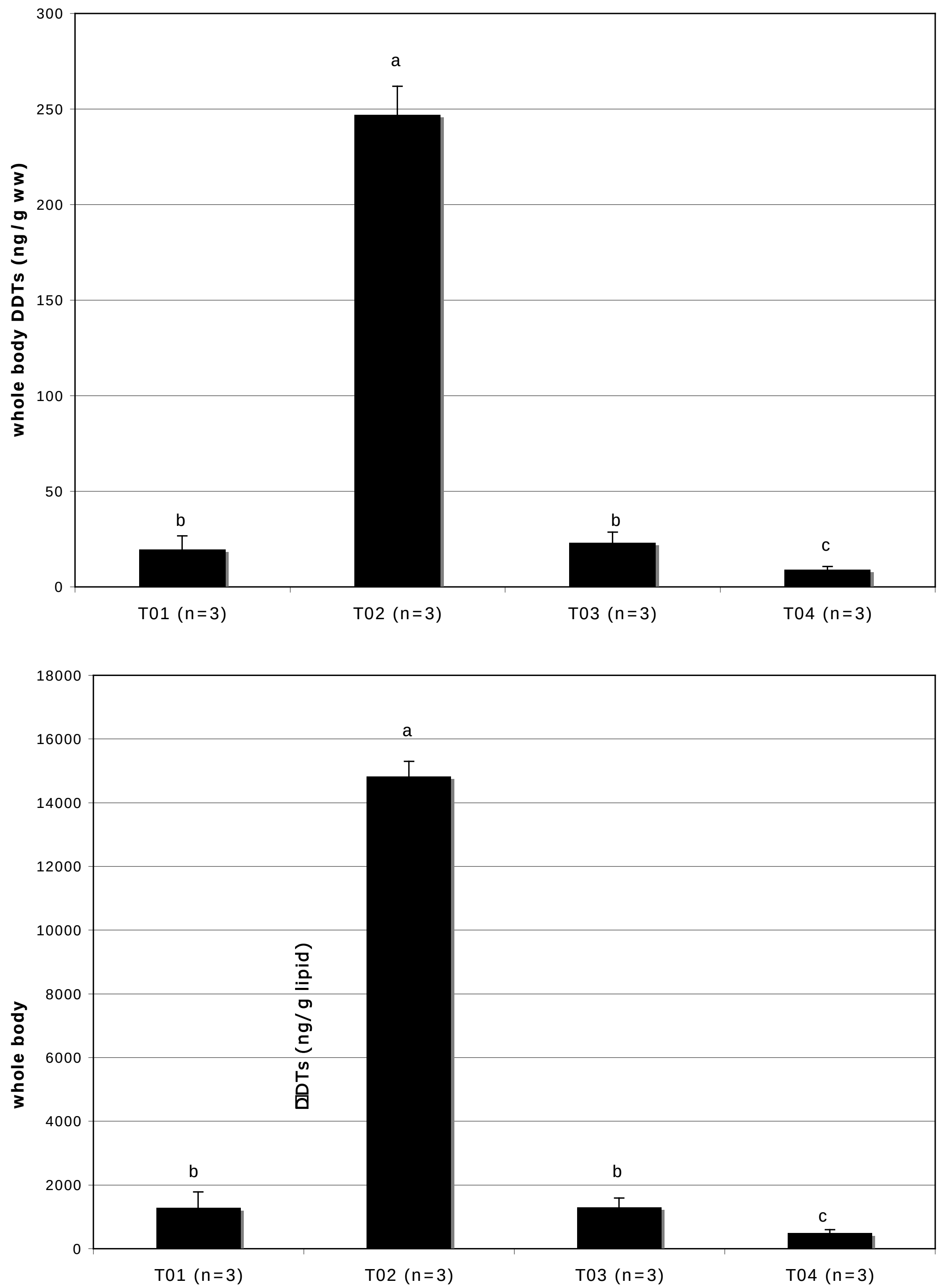


Figure 6. Concentrations of ∑DDTs (ng/g ww and ng/g lipid) in whole bodies of juvenile salmon from Willamette River sites. Mean values with different letter designations are significantly different (1-way ANOVA, Tukey-Kramer multiple range test, $p \leq 0.05$; log-transformed values).

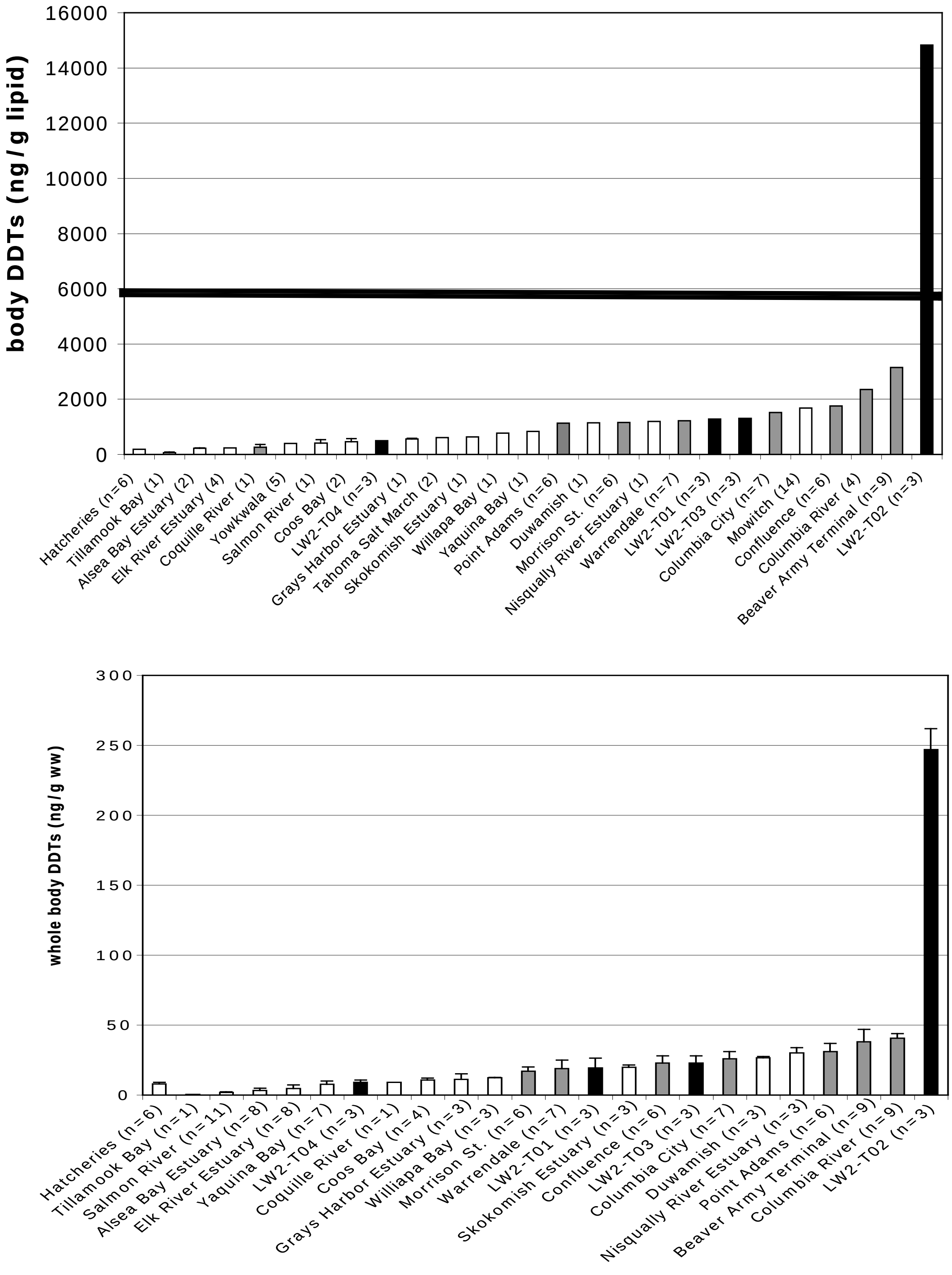


Figure 7. Concentrations of ∑DDTs (ng/g lipid and ng/g wet wt) in whole bodies of juvenile salmon from the Willamette River as compared other Pacific Northwest sites. Data from Johnson et al. 2006a,b; Leary et al. 2006). Solid line indicates estimated toxicity threshold, based on Beckvar et al. 2005.